D. Therapy of Acute Pulmonary Edema

I. Etiology of Pulmonary Edema

One of the major causes of pulmonary edema is acute left ventricular failure. Other types of pulmonary edema are also common. Severe pneumonia may be associated with pulmonary edema. The clinical picture may be identical to that of cardiac pulmonary edema. The ‘fluid lung’ of patients with renal failure is characterized by the retention of interstitial fluid. In the late stage of renal failure, the complete clinical picture of pulmonary edema might develop. Less common causes for pulmonary edema are exposure to toxic substances, nitric gases and narcotic overdose. The clinical findings of diffuse pulmonary hemorrhage in the Good-Pasture Syndrome can mimic the symptoms of pulmonary edema.

It is important for accurate therapy to determine the precise etiology of the pulmonary edema. Treatment that reduces filling pressure and reduces congestive symptoms makes sense only in cardiac pulmonary edema. In other forms of pulmonary edema, the alveolar-capillary membrane between the pulmonary alveolus and pulmonary capillary shows toxic damage. Slit-like openings develop through which fluid and proteins exude into the alveoli without being necessarily associated with an elevated pulmonary capillary wedge pressure.

In cardiac pulmonary edema, pressure elevation in the pulmonary capillaries causes fluid transudation into the alveoli. A pressure gradient of about 15 to 20 mmHg (filtration pressure) exists between the pulmonary capillaries and the alveoli. Between inspiration and expiration the alveolar pressure is ± 0 mmHg. The pulmonary capillary pressure has a normal value of up to 10 mmHg. If a filtration pressure of 15 to 20 mmHg is exceeded, that is an increase in pulmonary wedge pressure to 20 mmHg, water exudes into the alveoli. Below these values, fluid transudation is not possible since water is retained in the vessel by the colloid-osmotic pressure of proteins. The alveolar-capillary membrane represents another barrier.

The aim of various therapeutic regimens such as phlebotomy, nitroglycerin and diuretics is a rapid decrease in pulmonary capillary wedge pressure. Water passively rediffuses from the alveolus with reduction of this pressure (Riecker 1975).

In a few cases, particularly in juvenile patients with extensive infarction, pulmonary congestion can be demonstrated clinically and radiologically without evidence of pressure increases in pulmonary circulation. The cause of this non-hydrostatic-related pulmonary edema is unknown. A similar phenomenon has been demonstrated in animal experiments (Richeson et al. 1982).
II. Hemodynamics in Pulmonary Edema

Systematic hemodynamic measurements prior to initiating treatment for pulmonary edema are difficult to collect. Despite modern techniques and application of the Swan-Ganz floating catheter such measurements have rarely been described. The seven cases described by Bussmann et al. (1975) and Bussmann and Schupp (1977) are primarily patients with a right heart catheter in place who subsequently developed pulmonary edema.

These seven patients had a pulmonary artery systolic pressure ranging between 45 and 85 mmHg with a mean pressure of 63 mmHg. The pulmonary artery diastolic pressure corresponding to the left ventricular filling pressure was between 22 and 50 mmHg, with a mean of 33 mmHg. Cardiac output was depressed, and values between 2.1 and 4.2 l/min with a mean of 3.3 l/min were recorded. Relative to a body surface area of 1 m², this corresponds to a cardiac index of 2.0 l/min/m².

Arterial pressure values varied but were clearly elevated (mean value 153/95 mmHg). Arterial systolic pressures up to 200 mmHg were not rare. Some patients were found to have normal baseline values of blood pressure. The mean heart rate was 117 beats/min with fluctuations between 105 and 150 beats/min.

Magrini and Niarcos (1980) recorded similar hemodynamic values in patients with pre-existing chronic heart failure who developed acute left-sided heart failure with dyspnea, rales and orthopnea. The pulmonary capillary wedge pressure averaged 28 mmHg, cardiac output was 3.3 l/min and heart rate was increased to 102 beats/min. These patients also had right heart failure with elevation of the right atrial pressure to 28 mmHg.

III. Clinical Classification of Pulmonary Edema

Pulmonary edema of cardiac origin can be classified into four stages of increasing severity of clinical signs and symptoms (Bussmann and Schupp 1978):

Grade I: Pre-pulmonary edema
Mild pulmonary edema with dyspnea, mild wheezing, and minimal auscultatory moist rales.

Grade II: Pulmonary edema
Moderate edema with moderately clinically audible rales (at the mouth of the patient) and mild orthopnea.

Grade III: Severe pulmonary edema
Severe degree of pulmonary edema with orthopnea and clearly audible rales and rhonchi.

Grade IV: Very severe pulmonary edema, also called classical pulmonary edema.
The patient exhibits severe orthopnea and diaphoresis. Loudly audible rales and rhonchi.

About half of all infarction patients develop left ventricular failure. The severity of left-sided heart failure varies and may include pulmonary edema. In latent left heart failure associated with coronary artery disease, a superimposed minor infarction